Besides retrospective studies, recent prospective studies have also found an association of oral contraceptives and venous thrombosis.[4] Even the Walnut Creek study to which Dr Hougie refers found that oral contraceptive use was associated with an increased risk of idiopathic thromboembolism—that is, thrombosis without a recent surgical procedure, trauma, malignant condition or other known predisposing factor.[5] Some investigators feel that this evidence is weakened by the study's failure to find a statistically significant effect in patients predisposed to venous thromboembolism. They argue that an adverse effect of oral contraceptives should be most pronounced in patients at high risk for thrombosis. However, it can also be argued that other predisposing factors, when present, overwhelm the effect of oral contraceptives and prevent a small but real effect of those agents from being discernible.

In addition to the results of epidemiologic studies, much indirect evidence indicates an association of oral contraceptives and thromboembolic disease. High-dose, noncontraceptive estrogen therapy, such as for suppression of postpartum lactation, has been shown to increase the incidence of thromboembolism.[6] Furthermore, the decreased morbidity from venous thromboembolic disease in reproductive-age women that occurred coincidently with the introduction of contraceptive pills with reduced estrogen content[7] also implicates estrogen as a risk factor for deep venous thrombosis. Finally, the dose-related effect of oral contraceptives to create an apparent imbalance of the hemostatic mechanism towards hypercoagulability, while of uncertain physiologic significance, is consistent with the reported correlation between estrogen dose and the risk of venous thromboembolism.[8]

Thromboembolic events in all young women are infrequent, and so a conclusive answer regarding their incidence in oral contraceptive users as compared with nonusers is unlikely because of the enormous size required of a definitive study. While there is no undeniable proof for a causal relationship between oral contraceptives and venous thrombosis, there is a wealth of evidence for the association of the two which, after reviewing, I and many others have chosen to accept rather than disregard as the result of bias. Yet, I would emphasize that since the risks of oral contraceptive use are small and further reduced by minimizing the steroid content of the preparation used, oral agents are a wise choice for many women who want contraception.

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Relationship Between Costs and Quality of Medical Care

To the Editor: Your editorial in the August issue dealing with the measurement of quality and costs of medical care[1] reminded me of an interesting proposed relationship between the two. As a student several years ago in the Community Medicine program at the University of Utah School of Medicine I was exposed to the following equation:

$$\text{Quality of Medical Care} = \frac{\text{Outcome of Medical Care}}{\text{Cost of Medical Care}}.$$

Perhaps it was my undergraduate training in mathematics that predisposed me to "latch on" to this equation in such a complete way. Ever since, I have not been able to read an article or editorial dealing with costs and quality without thinking in terms of this relationship.

The beauty of such an expression is that it defines an admittedly "soft" or subjective variable (quality) in terms of two "hard" or more objective variables (outcome, cost). The outcome of a medical care event may be considered in terms of health or functional status, years of life saved or whatever. Costs may include direct costs only or might also include the indirect costs associated with the provision or consumption of that care. To the extent that one increases the value of the numerator while holding the denominator constant or, alternatively, decreases the denominator while holding the numerator constant, quality increases.

I must say, however, that I have always been troubled by my inability to "play" with this equation. For example, if one attempts to move cost from the denominator of the right-hand expression and place it in the lefthand expression what results is as follows: cost · quality = outcome. If one holds quality constant and increases cost, does outcome necessarily increase (improve)? Then if one wishes to move quality into the denominator of the right hand expression (by dividing each side by quality), what results is $\text{cost} = \frac{\text{outcome}}{\text{quality}}$. If one then holds outcome constant and increases quality does cost necessarily go down?

At any rate, given the importance of understanding the relationships between costs of medical care and quality of medical care, we might all benefit from adding this perspective to our conceptual armamentarium.

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Enterococcal Endocarditis After Sigmoidoscopy

To the Editor: The article by Rodriguez and Levine[1] in the June 1984 issue was timely, indeed. A patient of mine had just been admitted to Santa Monica Hospital with enterococcemia. She was a 67-year-old woman with a porcine mitral valve prosthesis for rheumatic heart disease, a pacemaker and multiple other medical problems.

In reviewing her history and clinical record, we realized she had undergone flexible sigmoidoscopy in December 1983 and did not receive antibiotic prophylaxis. Over the ensuing six months, the patient lost 5 kg (11 lb) of weight, had recurrent bouts of nausea and vomiting and had repetitive episodes of dizziness. Due to the patient's complex set of medical problems and multiple medications, consideration of the diagnosis of prosthetic valve endocarditis was not entertained until June 1984, at which time an astute resident drew blood specimens for culture. Two days later a heavy growth of enterococcus was identified in the blood and the patient underwent a six week course of intravenous antibiotic therapy. No other source for the enterococcemia could be elucidated.

While the 1977 American Heart Association (AHA) guidelines for prevention of bacterial endocarditis clearly recommend antibiotic prophylaxis for patients with prosthetic valves undergoing sigmoidoscopy,[2] a large percentage of endoscopists do not follow these guidelines.[3] Even in retrospect, the gastroenterologist who carried out this patient's sigmoidoscopy felt that prophylaxis was not indicated.

The case described above is at least the third now reported in the literature of enterococcal endocarditis following sigmoidoscopy. Perhaps all endoscopists and primary care physicians would do well to review the AHA guidelines for prophylaxis, particularly as screening flexible sigmoidoscopy is becoming more widespread in primary care offices.

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Methicillin-Resistant *Staphylococcus aureus*

TO THE EDITOR: We wish to report three cases of community-acquired methicillin-resistant *Staphylococcus aureus* (MRSA) infection in intravenous drug abusers. MRSA is a well-recognized nosocomial pathogen[1,2] and has been identified as a community-acquired pathogen among drug addicts in Detroit.[3] To our knowledge MRSA is not widely recognized as a cause of community-acquired disease in this area.

Reports of Cases

CASE 1. The patient, a 45-year-old drug abuser—a "skin popper"—was admitted with multiple cutaneous abscesses, from which grew MRSA (sensitive to chloramphenicol, gentamicin, trimethoprim-sulfamethoxazole and vancomycin). Although blood cultures were negative, a urine culture showed more than 10^5 colonies per ml of MRSA with identical sensitivities.

CASE 2. A 32-year-old woman was admitted with MRSA bacteremia (sensitive to chloramphenicol, gentamicin, trimethoprim-sulfamethoxazole, clindamycin and vancomycin). She was a heroin addict who said she had not shared needles. Three months previously she had been admitted to hospital for endocarditis due to a methicillin-sensitive *Staphylococcus aureus*.

CASE 3. A 33-year-old woman, a drug abuser, was admitted with chronic osteomyelitis of the symphysis pubis. Blood culture and biopsy of the site revealed MRSA with sensitivities identical to those in case 1. The patient denied drug injection in the groin region.

All three patients had a history of multiple admissions to hospital and frequent antibiotic administration before the admission during which MRSA was recovered.

Traditional empiric therapy for intravenous drug abusers with presumptive staphylococcal infections has included coverage with a penicillinase-resistant antibiotic, typically nafcillin or a first-generation cephalosporin. Our experience suggests that in a seriously ill intravenous drug abuser in whom *Staphylococcus aureus* is suspected, especially when there is a history of recent hospital admission or antibiotic use, empiric therapy should include vancomycin pending culture and sensitivity results.

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Reye's Syndrome and Salicylates

TO THE EDITOR: With regard to the role of salicylates in Reye's syndrome,[1] I would suggest an additional comment to Dr Trauner's summary recommendation "to try other means of reducing fever and discomfort in children with viral illnesses and, in particular, flulike illnesses and varicella . . . ." We should also be counseling parents as to the role of fever in the immunological response to infection and to decondition them from using antipyretics routinely when fever and discomfort are modest. My guess is that the association between Reye's syndrome and salicylates would not exist were it not for the large population of children who receive aspirin (and sometimes alternately acetaminophen) on a regular basis for any temperature elevation.

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